Greenlees (T. D.)

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CORTICAL PARALYSIS

WITH LOSS OF SPEECH.

By T. DUNCAN GREENLEES, M. B., Edin.,

Assistant Medical Officer to The Counties Asylum, Garlands, Carlisle, England.

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The literature bearing on the question of the localization of the functions of the brain is steadily accumulating, and any contribution to this subject is worthy of record both for purposes of comparison and for clinical investigation.

The following cases illustrate a form of paralysis always, or nearly always, associated with some derangement of the speech function. I am indebted to the records of this institution for the clinical and post mortem notes.

Case I.—*Mary Ann G.*, æt. 41, was admitted to Garlands Asylum on 20th June, 1884, suffering from dementia, with temporary outbursts of excitement. Her previous history was bad: she had served five years penal servitude, and was supposed to have led an immoral and dissipated life. There was no history of syphilis, but, two years previous to her admission, she had an apoplectic seizure, after which she became hemiplegic on the right side. On admission she was dull and much enfeebled mentally; she had a pinched expression and was quite incoherent in speech. The face was drawn to the right side, and she was paralyzed all

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down the same side—the paralysis being more marked in the right upper extremity, where the muscles were much wasted as compared with those of the opposite side. The pupils were unequal—the left being the smaller; the ordinary cutaneous reflexes were diminished on the right side, but the knee-jerk was increased on the same side of the body. Her tongue pointed slightly to the left side, but seemed perfectly mobile. There was ordinary dorsal spinal curvature, but at this time no active thoracic or abdominal disease was detected on examination.

In the early period of her residence in the asylum her vocabulary was limited to such expressions as "ah, ah," and "aye," but she understood perfectly what was said to her, and, later in her history she was able to articulate more complicated words, such as "oh dear," "eh," "ha, ha," and "tits," but she could neither write to copy nor was she able to recognize the letters of the alphabet.

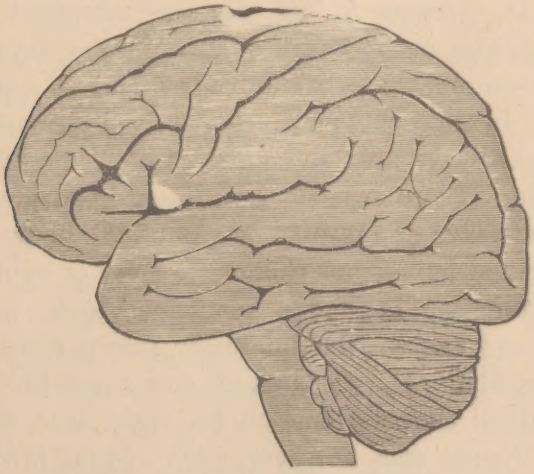
Several days after her admission she had an epileptiform seizure, the fit lasting several minutes, and after this she took an occasional fit—sometimes several in rapid succession—but the physical symptoms never seemed more pronounced after these attacks. Mentally she was dull and stupid, but extremely irritable and passionate, resisting care in any way.

During the winter of 1885–86 she became bedridden and developed symptoms of pulmonary tuberculosis to which she succumbed the following March, after a residence in the asylum of twenty-one months.

Autopsy.—The skull cap was thick and dense, but presented no local thickening, or nodular enlargements: the dura mater was slightly adherent to the vertex of the calvaria, and was thick and leathery in consistence. There was extensive effusion into the subarachnoid

space, especially over both parietal regions of the brain, and the cerebral convolutions were generally atrophied. The pia mater was slightly adherent to the subjacent gyri in localized patches: this condition was most marked over the left hemisphere in the immediate neighborhood of the fissures of Rolando and Sylvius.

On the left hemisphere the pia mater was much thickened, yellowish in color, and firmly adherent to the grey cortex to the extent of about a sixpenny piece



[FIG. 1.—MARY ANN G.—Left hemisphere, showing small area of softening.]

at the posterior termination of the third frontal convolution, just where that gyrus abuts on the Sylvian fissure, and immediately behind its horizontal branch. (See fig. 1.) Underneath this patch the brain tissue was softened and broke down easily to the depth of about three-fourths of an inch of the cerebral cortex.

The brain generally was softer than normal, and the left lateral ventricle was dilated, but no other pathological changes were observed in the contents of the cranial cavity.

The cortical lesion in this case was circumscribed to include the anterior portion of 9*, which, according to Ferrier, is the centre for the muscular movements of the mouth and tongue, (Aphasic centre), but as this patient was able to perform all necessary movements of the buccal and lingual muscles, such as in mastication, it is probable that the centre for motor impulses was intact, although the controlling and directing power—the centre for the transmission of idealized words to the organs of speech—in the cortex was impaired.

Case II.—*James K.*, æt. 53, was admitted to Garlands Asylum on January 8, 1885, having been insane for eighteen months previous, consequent on a paralytic seizure. In time the paralysis partly disappeared, but there had always been some degree of weakness on the right side. There was no hereditary history either as to the existence of insanity or any of the neuroses, and he never had had either rheumatic fever or syphilis.

On admission he had a suspicious look, was very restless, and had the fixed delusion that persons wished to poison him. He was a short and miserable looking man, walked slightly lame on the right side, the grasp of both hands was, however, about equal in strength. The cutaneous and tendon reflexes were normal, the pupils equal and contractile, and, with the exception of some slight intermittency in the heart's action and slight right-sided hemiplegia, no other physical disease was detected on his admission.

During his residence he was passionate and became easily excited; he had delusions as to where he lived, and would frequently shout out a few incoherent words. If left to himself he was dull and would speak to no one, preferring the solitude of his own company.

* The figures in this and the succeeding cases refer to those of Ferrier. See "Functions of the Brain," 2d ed. page 478.

His mental faculties gradually failed and he became more and more demented.

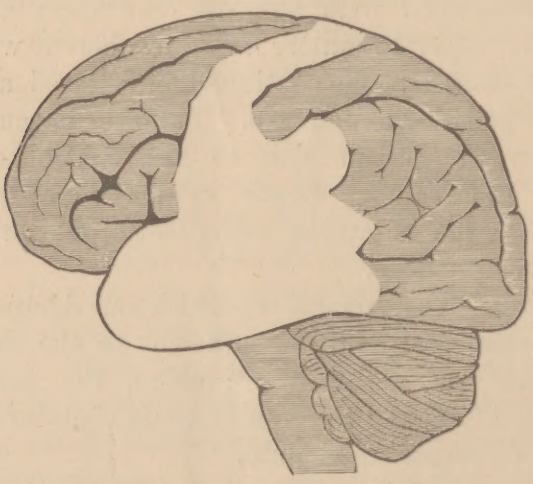
On July 1st, 1886, he had a paralytic seizure with convulsive movements of the right side. There was complete loss of power and sensation and the paralysis was accompanied with aphasia. Six days after the seizure he began to mutter a few incoherent words for the first time, but he continued in a dazed and only partially conscious condition. The skin began to give way over the sacrum and he lost control over the bladder and rectum. Death occurred nine days after the seizure, the breathing being stertorous for twenty-four hours previous.

Autopsy.—The calvaria was thick and dense, and the dura mater thin and not adherent to the skull cap. The brain was generally atrophied, the encephalon weighing 47 oz., and the cranial cavity contained several ounces of compensatory fluid.

Right hemisphere: The pia mater was not adherent. At the posterior border of the occipital lobe there was an area of cortical softening of about the size of one shilling, over which the meninges were raised in the form of a bulla, giving the portion implicated an œdematous appearance; this softening extended to the depth of about half an inch into the cortex of the brain. A small old hæmorrhage existed in the centre of the right corpus striatum.

Left hemisphere: On stripping the pia mater the following portions of the hemisphere were found lacerated; the ascending frontal convolution in its whole extent, the inferior portion of the ascending parietal convolution, the middle and inferior parietal convolutions and the whole of the temporo-sphenoidal lobe together with the Island of Reil. The temporo-sphenoidal lobe was in a state of red ramollissement

and broken down into a cavity, the limits of which could not be accurately defined. The cortical softening over the other portions of the hemisphere had destroyed the grey matter and encroached on the white but did not involve it to any great extent. (See fig. 2.)



[FIG. 2.—JAMES K.—Left Hemisphere, showing extensive cortical softening.]

The left hemisphere was somewhat softer and more congested than the right.

The heart was hypertrophied and the mitral valve very much constricted; the myocardium was healthy. Nothing worthy of note was observed in the other organs.

The above case is of interest from the extent of the cortical lesion, and goes far to verify the preconceived theories of the functions of the parts affected. A reference to Ferrier's diagram will show that the areas of softening in this case comprise 14 in its whole extent and the anterior portion of 13; 2, 3, 6, 7, 8, a portion of 9, the whole of 10 and 11, to a more limited extent. The more superficial lesions represent, according to Ferrier's experiments, the cortical centres for complex

movements of the opposite arm and leg as swimming or climbing, and movements of the opposite wrist and fingers as writing, together with movements of the mouth, tongue and lips (the aphasic centre).

With regard to the lesion of the temporo-sphenoidal lobe, Ferrier's experiments do not extend further than the superior convolution (14), but Nothnagel* thinks that "the connection between word-deafness and lesion of this convolution to be fairly well established;" Gowers† shows that irritating disease of the whole lobe may cause convulsions commencing with auditory aura, and it would seem, from the clinical history of this patient that he had hallucinations of the sense of hearing, which would explain the insane habit he had of occasionally shouting out a few incoherent words as if in reply to the voices he heard.

Case III.—*Hannah Eliza F.*, æt. 56, single, was admitted to this asylum on 18th May, 1880, suffering from mania probably due to the onset of the climacteric period. She had been insane for four months previous to her admission having delusions concerning her own personal identity, was eccentric in her habits and restless and sleepless at night.

On admission she was slightly exalted, restless and emotional, but perfectly coherent and her memory was fairly good. Physical examination revealed harsh breathing over both apices, an irregular and tumultuous action of the heart with a loud "booming" systolic murmur heard loudest over the mitral region, but propagated towards the axilla; the second sound of the heart was accentuated over the base.

There is little of importance to note for the first few

*"Regional Diagnosis of Diseases of the Brain," 1879, reviewed in "Brain," April, 1880, p. 98.

†"Diagnosis of Diseases of the Brain," 1885, p. 172.

years of her residence in the asylum; she suffered from chronic mania, but when her health permitted, she was able to do a little sewing occasionally. She was frequently under medicinal treatment for symptoms indicative of cardiac derangement; otherwise she enjoyed fairly good health for nearly five years.

On April 6th, 1885, she had an apoplectic seizure during which she was only partially conscious, and there was slight facial paralysis, the face being drawn to the left side, with ptosis of the left eyelid. She was able to protrude her tongue but it pointed to the right side, and her speech was somewhat impaired. Three days after the seizure the following note is recorded in her case: "She lies in a stupid and dazed condition but can be easily aroused; she is quite speechless and apparently unable to articulate the most simple words, although she understands perfectly all that is said to her. When asked to protrude her tongue she does so slowly and it points to the right side. There is complete right-sided hemiplegia, the right upper extremity is however most paralyzed; the mouth is drawn to the left side giving the right half of the face a characteristic expressionless appearance." During the next few months the paralysis gradually passed away leaving behind some weakness of the muscles of the right side. In time she was able to articulate a few words, mostly of one syllable, but when she attempted to carry on a conversation she merely uttered a long string of unintelligible and incoherent gibberish. Her mental condition became much worse; from being facile she became irritable, emotional, and at times even violent.

On December 26th she had a second and well-marked apoplectic seizure, and when examined she was profoundly comatose; both pupils were widely dilated

—the right most so—and neither responded to the influence of light; the various superficial and deep reflexes were totally abolished; the respirations were slow, prolonged, sighing and "cerebral" in character. Death occurred about seven hours after the onset of the attack.

Autopsy.—The skull cap was thick and the diploë were for the most part absorbed; the dura mater was partly adherent to the vertex of the calvaria; the meningeal vessels were full and injected, more especially over the occipital and more dependent regions of the brain. The arteries at the base and the internal carotids remained patent on section, were tortuous, and had numerous calcareous plates along their course. There was no adhesion of the pia mater to the subjacent convolutions. The left middle meningeal artery, as it lay within the Sylvian groove was blocked for nearly one inch of its extent with a partly organized clot, and the left temporo-sphenoidal and central lobes were softer than normal and than the remainder of the hemisphere, but there was no localized necrosis of brain tissue observed. Both halves of the cerebrum were soft on section, and the tissue of the lenticular nucleus of the left corpus striatum was of a creamy consistence and washed away under a gentle stream of water. In the superior portion of the medulla oblongata there was a small old hæmorrhage about one-third of an inch in extent.

The heart was large, weighing 14½ ounces, and the left ventrical hypertrophied. The aortic valve was incompetent, its cusps shriveled, and the mitral valve was very much constricted. The other viscera presented nothing worthy of note.

This case is an example of the more common types of cortical paralysis and aphasia due to an embolic plugging

of one of the main arteries of the brain; and, as the external or inferior frontal artery, a branch of the middle cerebral, is limited in its distribution to that region of the cerebrum more especially concerned in the complex function of speech, any interference to the circulation through the latter artery must necessarily produce aphasia. But, from the post-mortem appearance of the embolus, it was apparently of recent formation, and was presumably the primary cause of death, even before any consequent necrotic change took place in the parts supplied; we must, therefore, conclude that the lesion which, in the first place, caused the right-sided hemiplegia and aphasia eight months before death, was that situated in the upper portion of the medulla oblongata. A lesion in this position would intercept nerve impulses from the controlling centre in the cortex to the nerves, such as the hypoglossal and spinal accessory, which supply the muscles usually engaged in the process of articulation, and that, therefore, the lesion in this case was purely motor and not sensory.

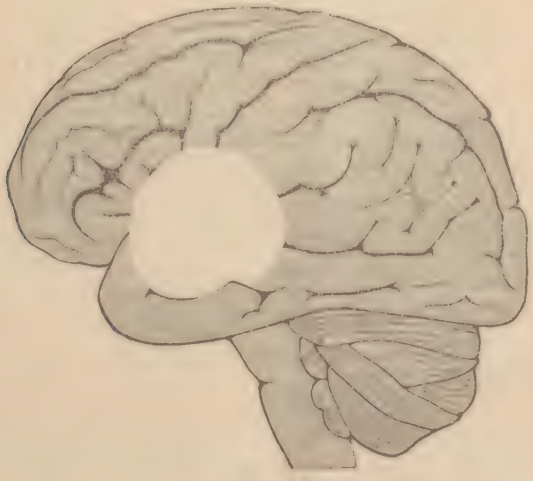
The softening of the lenticular body was probably of no importance in the production of the various symptoms.

Case IV.— *William B.*, æt. 32, was admitted from the workhouse to Garlands Asylum on June 28th, 1862. He was an imbecile, but liable to outbursts of violent excitement, and he was described as having been passionate if interfered with, but if left alone he would sit moping all day, and, in fact, led quite an automatic existence. His health was feeble, but no physical disease was detected on his admission.

For nineteen years he continued in much the same condition mentally, his bodily health improving somewhat, and he was able occasionally to assist at out-door employment.

In the early part of 1881 he developed symptoms of cardiac disease, and became subject to severe, but short attacks of cardiac asthma. In April of this year he had an attack of right-sided hemiplegia, but was not convulsed, and he did not lose consciousness. At the same time he became completely aphasic, and the power of speech never returned. Owing to the paralysis he was confined to bed; his heart symptoms became more urgent, dropsy supervened, and he died in the following January, (1882.)

Autopsy.—The pia mater was congested, especially over the left hemisphere, and there was general opacity of the meninges from subarachnoid effusion; the left half of the brain was atrophied and did not completely fill the skull cavity. There was a large patch of yellow



[Fig. 3.]

softening 4 inches by 3 inches in extent, involving the third or inferior left frontal, and the lower parts of the ascending parietal and ascending frontal convolutions, together with the anterior portions of the superior and middle temporo-sphenoidal convolutions, (see fig. 3);

this softened area comprised the whole of the Island of Reil and extended inwards to the corpus striatum, representing the portions of brain supplied by the left middle cerebral artery. The arteries on the left side were more atheromatous than those on the right, and the left hemisphere was nearly four ounces lighter than the right hemisphere.

The heart was hypertrophied and the mitral valve incompetent. This case is somewhat similar to the previous one, as in both it is apparent that the primary cause of the cortical softening was an arrest of the blood supply to a limited portion of the brain from an embolus set free from diseased cardiac valves.

The areas of brain affected in this case comprise 8, 9, 10, 11, the anterior portion of 13 with the corresponding part of 14; and, according to Ferrier's experiments, these parts represent centres for movements of the opposite upper extremity, especially the wrist and the hand, as in writing; various movements of the mouth, lips and tongue, as in speaking; the centres for hearing, and dilatation or contraction of the pupils.

The connection existing between agraphia and aphasia is full of instruction, and I offer no apology for reproducing here *in extenso* Dr. Gower's remarks with regard to those lesions—both sensory and motor—which comprise among their symptoms loss of speech and inability to write.*

"If the disease involves the motor paths some distance below the cortex, it may cause transient defect of speech; but this is soon recovered from, probably because the left region is able to act through the right by means of the commissural fibres of the corpus callosum. When the disease is just beneath the cortex these fibres are also damaged and the aphasia is as lasting as when the cortex itself is destroyed. In the extremely rare cases in which the patient can write and can not speak, the disease is probably so

* Diagnosis of Diseases of the Brain, 1885, pp. 130 and 131.

placed as to interrupt the fibres that go to the motor tract and those that go to the corpus callosum, but has not destroyed the speech centre itself or the connection between it and the hand centre."

In cases where the lesion is extensive not only is the power of speech lost but there is also agraphia, even although there is no paralysis of the right hand. In writing the words are first evolved in the speech centre, they then pass to the hand centre, and thence along the motor fibres to the nerves which supply the muscles necessary for the process. If the disease, however, is persistent and extensive the right hemisphere may be so educated that the impulse is evolved from it in time; thus in both Cases I and III a certain amount of improvement in the power of articulating various words took place, and, as the lesion in both was persistent, it is highly probable that the corresponding parts of the cortex of the right hemisphere underwent a certain amount of education for the purpose of discharging the functions of the damaged portions of the left hemisphere. It is interesting, in connection with this subject, to note that in children this power of educating one hemisphere to discharge the duties of the other, is stronger than in adult life or old age.